Clinical Symptoms in Patients With Sustained Ventricular Tachycardia

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We questioned 113 patients with subsequently diagnosed sustained ventricular tachycardia (VT) regarding the symptoms that prompted their seeking hospital treatment, eliciting the following: 15% of patients had lost consciousness, 15% had near syncope, 35% had mild lightheadedness and 35% had no cerebral symptoms. Patients with preexisting congestive heart failure or a VT rate of 200 beats per minute or greater more often lost consciousness. Other symptoms included palpitations in 57% of patients, chest pain in 27%, dyspnea in 25%, weakness in 6%, nausea or diaphoresis in 3% each and flushing in 2%. In approximately 50% of patients who had mild lightheadedness or no cerebral symptoms, their condition was incorrectly diagnosed as supraventricular tachycardia based on the absence of severe symptoms during the tachycardia. In some patients, VT may be associated with mild or atypical symptoms. The differentiation of supraventricular from ventricular tachycardia should be based on electrocardiographic criteria and should not be influenced by the nature or severity of a patient's symptoms. The severity of cerebral symptoms is at least partially related to the VT rate and a patient's underlying heart disease.

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Ventricular tachycardia (VT) is a potentially fatal arrhythmia and is the second most common cause, after ventricular fibrillation, of out-of-hospital cardiac arrest.[1-5] Ventricular tachycardia has typically been described as a serious arrhythmia that is nearly always associated with severe symptoms such as loss of consciousness or seizures and nearly always requires emergent treatment, especially when it occurs in patients who have structural heart disease and when the VT rate is rapid (200 beats per minute or more).[6-11] Perhaps less well appreciated is that VT need not result in severe hypotension or shock. Although there have been several reports of patients who had mild symptoms associated with VT, these reports have been based on small numbers of patients, many of whom were young and had no apparent structural heart disease.[12-20]

In our experience, a significant proportion of patients presenting with sustained VT may not have severe symptoms requiring emergent treatment, even when the VT rate is rapid and the patient has structural heart disease. We have frequently noted cases in which VT was incorrectly diagnosed as supraventricular tachycardia because of a patient's mild symptoms. We describe herein the clinical symptoms in a large group of patients who sought hospital treatment for what proved to be sustained VT. Our observations show that VT may be associated with a wide range of symptoms that are not always severe or typical.

Methods

We interviewed 113 patients who were referred to our institution for management of VT. All had sought hospital treatment for what proved to be sustained VT of at least 30 minutes' duration. Patients who had VT associated with out-of-hospital cardiac arrest were excluded from this series, as were patients who had VT with acute myocardial infarction. In addition, patients with a known history of VT for which they were being treated with antiarrhythmic drugs were not

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included. In all cases, a wide-complex tachycardia was documented by an electrocardiogram, which was available for review. In 12 patients, the diagnosis of VT was based on the presence of independent atrial activity or fusion beats (or both) on the electrocardiogram recorded during the tachycardia. In the remaining 101 patients, clinical tachycardia was provoked during electrophysiologic testing by programmed ventricular stimulation and was shown to be ventricular in origin based on either the presence of atrioventricular dissociation or the absence of a His bundle depolarization preceding the ventricular depolarizations (or both of these) and the response to atrial and ventricular pacing during the tachycardia.

Patients were interviewed by one or more of the authors within a month of the episode of spontaneous VT and were questioned regarding their symptoms during VT. The patients were specifically questioned with regard to cerebral symptoms, chest pain, dyspnea and palpitations. In addition, histories obtained by physicians during the episodes of VT were reviewed to check for any discrepancies with the patients' responses.

All patients were examined by at least one of the authors. A diagnosis of congestive heart failure was based on the presence of a third heart sound gallop and a radiographic demonstration of cardiomegaly and pulmonary venous congestion.

Clinical Profile

The mean age of the patients was 57 ± 15 years. In all, 81 had coronary artery disease (with a history of myocardial infarction in 72), 10 had hypertensive heart disease, 7 had a congestive cardiomyopathy, 2 had isolated mitral valve prolapse, 1 had aortic regurgitation and 1 had undergone surgical repair of a tetralogy of Fallot. Eleven patients had no evidence of structural heart disease, with no abnormalities detected during physical examination, two-dimensional echocardiography or exercise treadmill testing.

Congestive heart failure was present in 58 patients (51%); 68 patients were being treated for supraventricular arrhythmias or ventricular premature depolarizations with quinidine sulfate, procainamide hydrochloride or disopyramide phosphate at the time they sought hospital treatment for VT, and 26 patients were being treated with propranolol hydrochloride for angina pectoris.

Statistical Analysis

Values are expressed as mean ± one standard deviation. The mean VT rates among the patients with syncope, near syncope, lightheadedness and no symptoms of cerebral hypoperfusion were compared using a one-way analysis of variance, with Cheffe's method for multiple contrasts. Other comparisons were done using either Student's t test or χ^2 analysis.

Results

The mean VT rate was 179 ± 37 beats per minute. There was no significant difference in the VT rate between patients who were and were not taking antiarrhythmic drugs (175 ± 37 beats per minute and 186 ± 36 beats per minute, respectively; $P > .05$).

Symptoms Related to Cerebral Hypoperfusion

In all, 17 patients had loss of consciousness, either persistent (10) or transient (7), in association with VT of a mean rate of 224 ± 29 beats per minute. Another 17 patients experienced near syncope and had a mean VT rate of 191 ± 38 beats per minute. Lightheadedness without syncope or near syncope was present in 40 patients in association with a mean VT rate of 170 ± 26 beats per minute. Of the 113 patients, 39 (35%) said they had no symptoms of lightheadedness, near syncope or syncope and they had a mean VT rate of 163 ± 31 beats per minute. A comparison of the VT rates among these groups of patients is shown in Table 1. The mean VT rate in the patients who had syncope was significantly greater than in the other groups of patients. In addition, patients with near syncope had a mean VT rate that was higher than that in patients who had no symptoms of cerebral hypoperfusion.

Patients with congestive heart failure had a significantly higher incidence of syncope than patients without congestive heart failure (Table 2). Within the group of patients who had syncope, there was no significant difference in the mean VT rate between patients with or without congestive heart failure. Patients without congestive heart failure more often had no symptoms of cerebral hypoperfusion than did those with congestive heart failure.

In comparing 35 patients who had a VT rate of 200 beats per minute or greater with 78 patients who had a VT rate of

TABLE 1.—*Cerebral Symptoms in 113 Patients Who Had Sustained Ventricular Tachycardia (VT)*

Symptom	Patients Number (%)	VT Rate Beats/min*	P Values†
A Syncope	17 (15)	224 ± 29	A v B < .05 A v C < .001 A v D < .001
B Near syncope	17 (15)	191 ± 38	B v C NS B v D < .05
C Lightheadedness	40 (35)	170 ± 26	C v D NS
D None of the above	39 (35)	163 ± 31	

v = versus

*Mean ± one standard deviation.

†NS = not significant ($P > .05$).

TABLE 2.—*Cerebral Symptoms During Ventricular Tachycardia in 55 Patients With and 58 Patients Without Congestive Heart Failure (CHF)*

Symptom	Patients With CHF Number (%)	Patients Without CHF Number (%)
Syncope	13 (24)	4 (7)*
Near syncope	6 (11)	12 (21)
Lightheadedness	21 (38)	19 (33)
None of the above	6 (11)	32 (55)†

*$P < .05$ (comparison of incidence of syncope among patients with and without CHF).

†$P < .001$.

TABLE 3.—*Cerebral Symptoms During Ventricular Tachycardia in 78 Patients With Heart Rate of Less Than 200 Beats per Min and 35 Patients With Heart Rate of 200 Beats per Min or More*

Symptom	Rate < 200 Beats/Min Number (%)	Rate ≥ 200 Beats/Min Number (%)
Syncope	4 (5)	13 (37)*
Near syncope	8 (10)	10 (29)†
Lightheadedness	32 (41)	8 (23)
None of the above	34 (44)	4 (11)*

*$P < .005$.

†$P < .05$ (comparison of patients with rate < 200 beats/min versus those with rate ≥ 200 beats/min).

less than 200 beats per minute, the former more often experienced syncope or near syncope and less often had no symptoms of cerebral hypoperfusion than the latter (Table 3).

Other Symptoms During Ventricular Tachycardia

Chest pain during VT occurred more often in patients with coronary artery disease than in those without coronary artery disease (33% versus 12%, $P < .05$) (see Table 4). The incidence of dyspnea during VT was not significantly greater in patients who had congestive heart failure than in those who did not (33% versus 17%, $P > .05$).

Eight patients did not have symptoms of cerebral hypoperfusion or palpitations during VT. Five of the eight complained only of chest pain, one had shortness of breath, one only nausea and epigastric discomfort and one described his only symptom as being epigastric "fullness."

Illustrative Cases

CASE 1. A 34-year-old woman who had mitral valve prolapse had noted two episodes of syncope over a period of a year. On the day of admission to hospital, she experienced an abrupt loss of consciousness for 10 to 15 seconds followed by a persistent sensation of near syncope. When she arrived at the emergency room, her blood pressure was 80/50 torr and heart rate 250 beats per minute. She said she did not have chest pain, dyspnea or palpitations. An electrocardiogram showed a wide-complex tachycardia at a rate of 250 beats per minute, which was terminated with direct-current countershock. The tachycardia was later provoked by programmed ventricular stimulation and was shown to be VT.

Comment. This case illustrates that sustained VT can cause a transient loss of consciousness, probably by initially causing an abrupt drop in blood pressure that is then partially reversed by reflex vasoconstriction. Another possible explanation in this patient is that the VT rate was initially faster than 250 beats per minute. Although her previous episodes of syncope and near syncope were in all likelihood caused by VT, she said she had no sensation of palpitations before or during the episodes. Therefore, the lack of palpitations does not exclude the possibility that cerebral symptoms are related to VT.

CASE 2. The patient, a 64-year-old man with a history of myocardial infarction and congestive heart failure, came to the emergency room after having palpitations, mild shortness of breath and intermittent mild chest pain for two hours. His heart rate was 220 beats per minute and blood pressure 100/60 torr. He said he did not have any cerebral symptoms. An electrocardiogram showed a wide-complex tachycardia at a rate of 220 beats per minute, with atrioventricular dissociation. Carotid sinus massage was unsuccessful in terminating the tachycardia. The patient was then treated with intravenous administration of 10 mg of verapamil. Shortly thereafter, his blood pressure fell to 70 torr systolic, and he complained of near syncope and chest pain. The tachycardia was then terminated with direct-current countershock. During electrophysiologic testing, the tachycardia proved to be ventricular in origin.

Comment. The tachycardia was incorrectly diagnosed as supraventricular because of the patient's mild symptoms and well-maintained blood pressure. About 50% of patients in this series who presented with mild lightheadedness or no cerebral symptoms during VT were also incorrectly diagnosed as having supraventricular tachycardia for similar reasons. This led to inappropriate treatment with drugs such as verapamil, which in this case resulted in severe hypotension. This case shows that VT may at times be associated with mild symptoms even when the rate is more than 200 beats per minute and the patient has underlying congestive heart failure.

CASE 3. The patient, a 62-year-old man with a history of myocardial infarction, had noted several episodes of nausea and epigastric discomfort lasting from 30 minutes to 3 hours over a period of six months. On one occasion, these symptoms persisted for several hours, at which time he went to a hospital. His heart rate was 160 beats per minute and blood pressure 110/60 torr. He said he had no symptoms other than nausea and epigastric discomfort. An electrocardiogram showed VT at a rate of 160 beats per minute. His symptoms of nausea and epigastric discomfort resolved after termination of VT by intravenous administration of lidocaine. When later undergoing electropharmacologic testing, his typical symptoms of nausea and epigastric discomfort were reproduced each time clinical VT was provoked by programmed ventricular stimulation.

Comment. This case shows how VT may at times cause atypical symptoms, in this case gastrointestinal symptoms.

TABLE 4.—*Symptoms (Other Than Cerebral) Experienced by 113 Patients During Sustained Ventricular Tachycardia**

Symptom	Patients Number (%)
Palpitations	64 (57)
Chest pain	31 (27)
Dyspnea	28 (25)
Weakness	7 (6)
Nausea	3 (3)
Diaphoresis	3 (3)
Flushing	2 (2)

*Some had more than one symptom.

Discussion

Although VT may often be a life-threatening arrhythmia requiring emergent treatment, the present series of selected patients who sought hospital treatment for what proved to be sustained VT shows that VT may be associated with a wide range of symptoms that are mild to severe. In this series, 69% of patients noted only mild lightheadedness or no cerebral symptoms at the time of hospital admission. About 50% of these patients were incorrectly diagnosed as having supraventricular tachycardia based on the absence of severe symptoms during the tachycardia. In several cases, this incorrect diagnosis led to inappropriate therapy with drugs aimed at the treatment of supraventricular tachycardia, such as digoxin or verapamil. In addition to delaying the onset of appropriate therapy, treatment with drugs such as verapamil may result in severe hypotension when administered intravenously to patients with VT. It is therefore important to recognize that VT may at times be associated with mild symptoms. The differentiation between supraventricular and ventricular tachycardia should be based on electrocardiographic criteria, not on whether or not a patient has severe symptoms during the tachycardia.

The severity of cerebral symptoms was at least partially

related to the rate of the VT—that is, patients with syncope had a higher mean VT rate than other patients and patients with near syncope had a higher mean VT rate than patients with no cerebral symptoms. In addition, patients who had a mean VT rate of 200 beats or more per minute had a 65% incidence of syncope or near syncope compared with only 15% among patients whose VT rate was less than 200 beats per minute. Nevertheless, it should be noted that 34% of patients who had a VT rate of 200 beats or more per minute had only lightheadedness or no symptoms of cerebral hypoperfusion during the VT. Patients with underlying congestive heart failure had a higher incidence of syncope during VT than did patients without congestive heart failure. This was the case even though there was no difference in the mean VT rate between these two groups of patients. Therefore, as would be expected, patients with depressed left ventricular function are less able to tolerate a given rate of VT.

Chest pain and dyspnea were each present in about a fourth of patients with VT. As might be expected, chest pain was more common in patients with known coronary artery disease. Although there was a tendency for dyspnea to be more frequent among patients with underlying congestive heart failure than in those without heart failure, the difference was not statistically significant.

Palpitations are often regarded as a classic symptom of paroxysmal tachycardia. In one report of 120 patients with paroxysmal tachycardia (supraventricular in 113), only 7% did not have palpitations.[21] However, in our series about a third of the patients with sustained VT did not have palpitations during the tachycardia. This observation has important implications regarding the evaluation of patients with unexplained syncope or near syncope. The lack of palpitations in association with these symptoms does not exclude the possibility that the symptoms are due to VT.

It is interesting to note that two patients had no palpitations, dyspnea, chest pain or cerebral symptoms during VT but complained of nausea or epigastric discomfort or both. Such symptoms in association with VT have been reported in a few patients.[15] Therefore, VT may rarely present with symptoms that are more gastrointestinal than cardiovascular.

In conclusion, it should be emphasized that the patients in this series are not a representative sample of all patients who have VT. In many patients VT may result in cardiac arrest and death. It is not our wish to minimize the malignant potential of VT but rather to highlight the observation that in a selected subgroup of patients who arrive at hospital with sustained VT that is not associated with cardiac arrest or acute myocardial infarction, it may be associated with relatively mild symptoms. The differentiation of supraventricular from ventricular tachycardia should be based on electrocardiographic criteria[22] and should not be influenced by whether a patient's symptoms are mild or severe.

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